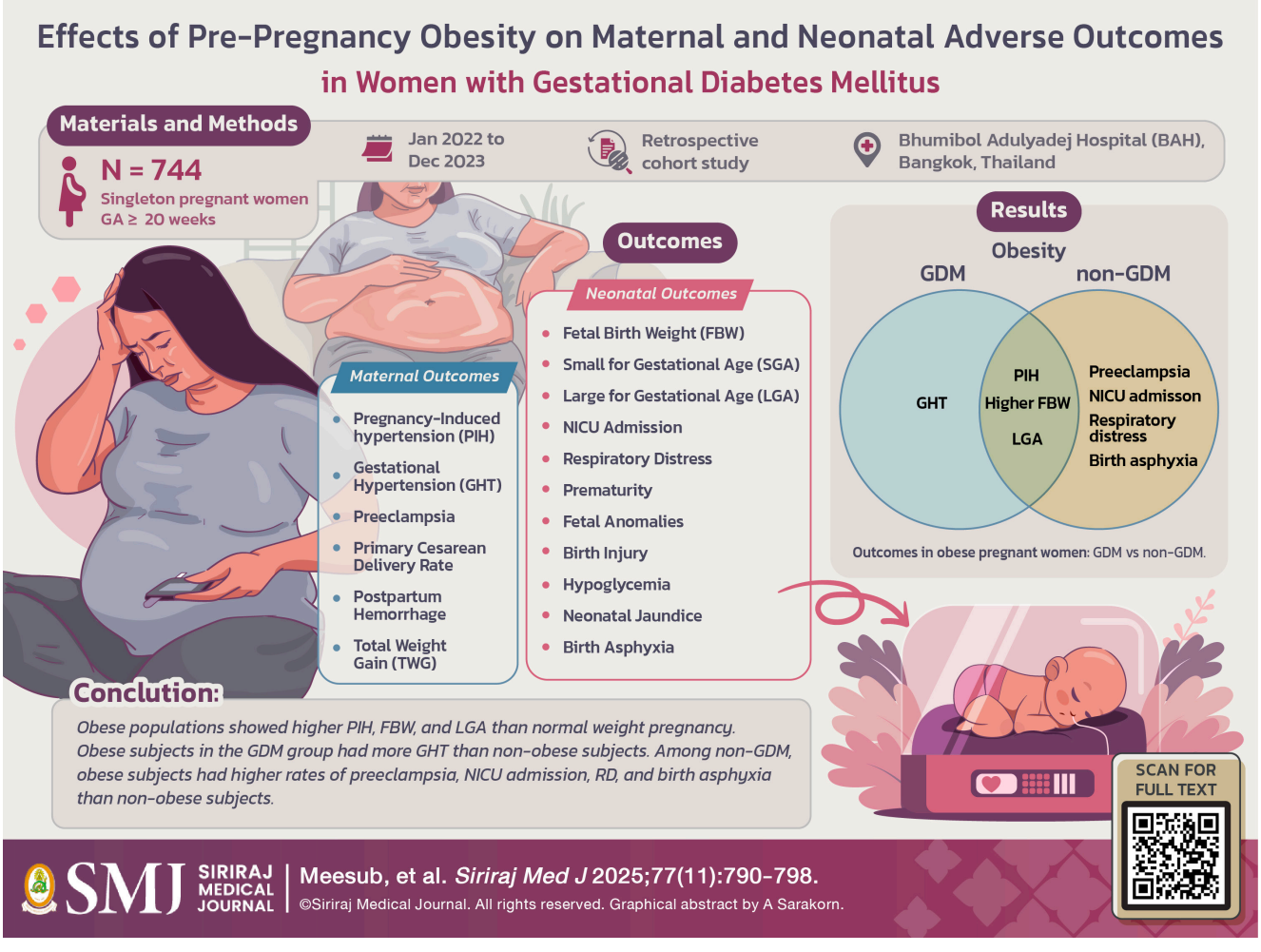
Effects of Pre-Pregnancy Obesity on Maternal and Neonatal Adverse Outcomes in Women with Gestational Diabetes Mellitus

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ABSTRACT

Objective: To explore the effects of pre-pregnancy obesity and GDM on pregnancy outcomes.

Materials and Methods: This retrospective cohort study was conducted between January 2022 and December 2023 at Bhumibol Adulyadej Hospital (BAH), Bangkok, Thailand. Participants were singleton pregnant women aged between 16 and 45 years old and delivered at BAH. Exclusion criteria were prepregnant medical diseases. All subjects underwent a 75-g oral glucose tolerance test for diagnosis of GDM. There were GDM and the non-GDM groups. Data was reviewed from electronic medical records, including demographic and clinical characteristics; namely: total weight gain (TWG), pregnancy induced hypertension (PIH), fetal birth weight (FBW), LGA neonates, neonatal intensive care unit (NICU) admission, mild respiratory distress (RD), and birth asphyxia.

Results: A total of 744 participants were included and randomly divided equally into GDM and non-GDM groups, with 372 cases each. PIH, FBW and LGA of obese subjects in both GDM and non-GDM groups were significantly higher than those in non-obese subjects. Subjects with obesity in both groups had significantly less TWG than those in non-obese subjects. GDM group participants with obesity had a significantly higher prevalence of Gestational hypertension (GHT) than non-obese subjects. Among the non-GDM group, obese subjects had significantly higher prevalence of preeclampsia, NICU admission, mild RD, and birth asphyxia than non-obese subjects.

Conclusion: Obese populations showed higher PIH, FBW, and LGA than normal weight pregnancy. Obese subjects in the GDM group had more GHT than non-obese subjects. Among non-GDM, obese subjects had higher rates of preeclampsia, NICU admission, RD, and birth asphyxia than non-obese subjects.

Keywords: Obesity; pregnancy outcomes; gestational diabetes mellitus (Siriraj Med J 2025; 77: 790-798)

INTRODUCTION

Gestational diabetes mellitus (GDM) is a temporally abnormal glucose metabolism during pregnancy.[1] Insulin resistance and inadequate insulin response occur among GDM subjects.[2] Moreover, fetal hyperglycemia develops following maternal hyperglycemia. Sustained high blood sugar in the fetus induces hyperinsulinemia and stimulates excessive somatic fetal growth, causing macrosomia and large-for-gestational age (LGA) neonates. Fetal macrosomia is defined as fetal birth weight exceeding 4,000 g or 4,500.[3] LGA neonates are defined as fetal birth weight of more than the 90th percentile. Fetal macrosomia and LGA lead to various consequences, such as an increased rate of cesarean delivery and shoulder dystocia at birth.[4]

GDM and obesity among pregnant women are the most common metabolic complications in pregnancy. Obese pregnant women have increased insulin resistance and uncontrolled weight gain.[5,6] Increased risk of macrosomia, higher rate of cesarean delivery due to labor dystocia[7], maternal perioperative complications and neonatal sepsis were reported among obese pregnant women with normoglycemia.[8-10] Obesity is defined according to the definition by the World Health Organization Asian Pacific region as a body mass index (BMI) threshold of more than or equal to 25 kg/m^2,[11] while in the U.S., BMI equal or more than 30 kg/m^2 is used.[12]

Previous studies reported that maternal obesity at the first antenatal care (ANC) was associated with a higher neonatal birth weight, macrosomia's risk, and cesarean delivery rate than those with GDM.[13-16]

The aim of the study was to investigate the effects of pre-pregnancy obesity and GDM on neonatal birth weight, macrosomia, cesarean delivery rate, low Apgar score, neonatal intensive care unit (NICU) admission, and other adverse pregnancy outcomes among pregnant Thai women.

MATERIALS AND METHODS

Study design and setting

The retrospective cohort study was conducted at the Department of Obstetrics and Gynecology, Bhumibol Adulyadej Hospital (BAH), Bangkok, Thailand. It was approved by the Institutional Review Board of BAH (IRB 77/66), Royal Thai Air Force.

Study participants and data collection

The subjects included pregnant women who visited the antenatal clinic and delivered their children at BAH. Data were reviewed from medical records kept in BAH computerized databases between January 2022 and December 2023. The inclusion criteria included healthy women carrying singleton pregnancies with gestational age (GA) ≥ 20 weeks. Exclusion criteria included women who had pre-existing underlying diseases, namely Diabetes mellitus (DM), Chronic hypertension (CHT), and antiphospholipid syndrome (APS).

Demographic characteristics, height, and weight were gathered during the first antenatal visit. Maternal BMI was calculated as weight (kg) divided by height squared (m^2). According to the Asia-Pacific criteria, BMI over 25.0 kg/m^2 was diagnosed as maternal obesity.[11] All participants underwent GDM screening between 24 and 28 weeks of gestation.

A 75-g oral glucose tolerance test (OGTT) was the first step for screening GDM. From IADPSG criteria, GDM was diagnosed when any blood glucose level was above the cut-off values of fasting plasma glucose (92 mg/dL), 1-hr glucose (180 mg/dL), or 2-h glucose (153 mg/dL).[17]

All pregnant women were followed up by obstetricians until delivery. The non-GDM pregnant women were monthly appointed in the first and second trimester. Appointments every two weeks were applied during the third trimester of pregnancy. Weekly antenatal visits were appointed for women with GDM. In addition, consultation to endocrinologists, nutritionists, and dieticians was applied for pregnant women with GDM.

To ensure adequacy of glycemic control, Self-monitoring of blood sugar was regularly performed among pregnant women with GDM. When the glycemic target was not achieved, insulin was prescribed. Targets for glycemic control were recommended for fasting 1-hr and 2-hr postprandial glucose values were less than 95 mg/dL, 140 mg/dL and 120 mg/dL, respectively.[18] Women in the non-GDM group were followed up the same in either the obesity group or non-obesity group.

Subjects were divided into the study (GDM) and the control (non-GDM) groups. Comparisons were made between obese and non-obese subjects in both groups regarding clinical and adverse pregnancy outcomes. Maternal outcomes consisted of pregnancy-induced hypertension (PIH), gestational hypertension (GHT), preeclampsia, primary cesarean delivery rate, and postpartum hemorrhage.

The fetal outcomes included fetal birth weight (FBW), large-for-gestational age (LGA) neonate (weight over the 90th percentile for gestational age), and small-for-gestational age (SGA) neonates (weight below 10th percentile for gestational age), fetal macrosomia, preterm birth (below 37 weeks of gestation), NICU admission, respiratory complication, fetal anomaly, hypoglycemia (serum glucose concentration < 40 mg/dL in symptomatic, or < 45 mg/dL in asymptomatic term neonates), neonatal jaundice and birth asphyxia (an Apgar score of <7).

Statistical analysis

All statistical analyses were performed using Stata Statistical Software: Release Version 16. All statistical analysis involved the chi-square, independent T-test. A p-value of < 0.05 was classified as statistically significant.

RESULTS

Medical records of 4,694 pregnant women who visited the antenatal clinic and delivered their children at BAH between January 2022 and December 2023 were retrieved. Inclusion criteria were singleton pregnant women, 20 weeks of gestation or more and delivery at BAH. Exclusion criteria were maternal medical conditions that affected pregnancy outcomes, namely chronic hypertension, diabetes mellitus (DM) and antiphospholipid syndrome (APS).

As shown in Fig 1, 744 subjects were recruited for analysis. All subjects were divided into GDM and non-GDM groups, each with 372 cases, respectively. There were 186 obese and 186 non-obese women in the GDM group. There were 152 obese and 220 non-obese women in the non-GDM group.

The mean age of all subjects was 30.6 years old. Obese subjects in both groups had higher BMI and pre-pregnancy weights (PPW) than non-obese counterparts with statistical significance as shown in Table 1. Only the TWG of obese subjects were lower than those non-obese subjects with statistical significance. The average GA of obese and non-obese subjects among the non-GDM group was 38.9 and 38.6 weeks (p=0.026), respectively. Average GA among obese and non-obese members in the GDM group were comparable (38.1/38.1 weeks).

From Table 2, mean fetal birth weight (FBW), LGA and pregnancy induced hypertension (PIH) prevalent in obese pregnant women in both GDM and non-GDM groups were significantly higher than in normal-weight pregnant women. Among GDM pregnancies, obese pregnant women had a higher rate of GHT development than non-obese subjects with statistical significance. However, among the non-GDM group, obese pregnant women had higher rates of preeclampsia, respiratory distress syndrome (RDS), NICU admission, and birth asphyxia than those of normal-weight pregnant women as presented in Table 2.

DISCUSSION

Obesity was diagnosed using BMI values. According to WHO guidelines, BMI of equal to or more than 30 kg/m^2 was classified as obesity. Data from the Asia-Pacific study, cut points of BMI for obesity of people in the region was lowered to 25 kg/m^2.[19] From the current study, TWG of obese pregnant women was significantly less than those of non-obese pregnant subjects. Ke and

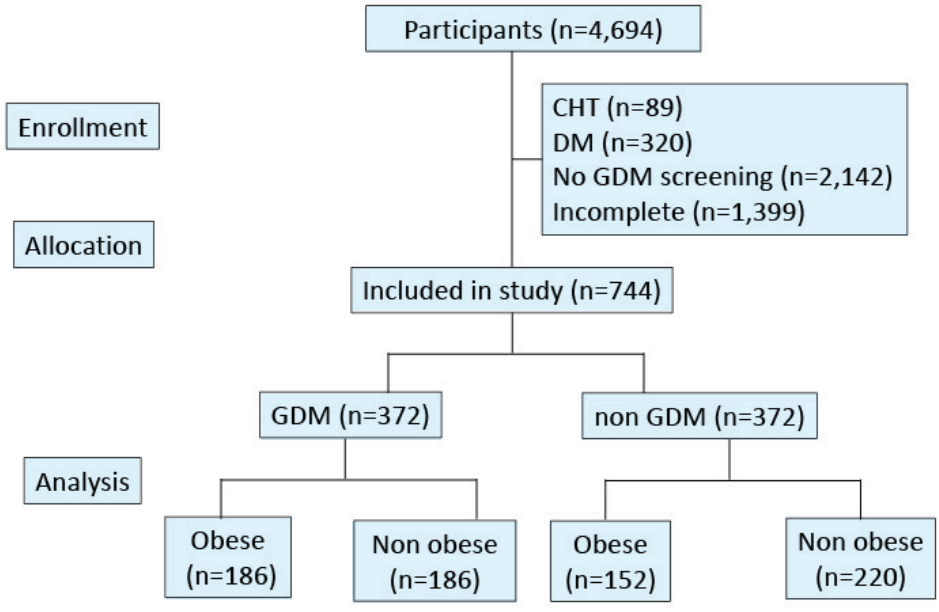


Fig 1. Flow chart of study.
Abbreviations: GDM: gestational diabetes mellitus, CHT: chronic hypertension

TABLE 1. Maternal baseline characteristics and maternal and fetal outcomes by gestational diabetes mellitus (GDM) and obesity status (n=744).

	GDM (n=372)*		*p*-value	Non-GDM (n=372)*		*p*-value
	Obesity (186)	Non (186)		Obesity (152)	Non (220)	
Age (yr)**	31.4±5.1	31.7±5.9	0.533	30.5±5.5	29.1±5.8	0.017
PPW (kg)**	75.6±11.5	52.6±7.5	<0.001	76.0±11.6	53.6±7.4	<0.001
Height (cm)**	158.2±6.2	157.5±6.3	0.243	159.9±5.94	159.7±5.8	0.750
PPBMI (kg/m^2)**	30.1±3.79	21.2±2.47	<0.001	29.7±3.94	21.0±2.3	<0.001
TWG (kg)	10.3±5.8	12.8±5.7	<0.001	13.6±7.2	15.3±5.8	0.015
Smoking	0 (0)	3 (1.6)	0.082	1 (0.7)	1 (0.5)	0.792
GA (weeks)**	38.1±1.7	38.1±1.2	0.859	38.9±1.2	38.6±1.4	0.026
Underlying disease						
Thyroid disease	3 (1.6)	3 (1.6)	1.000	2 (1.3)	3 (1.4)	0.969
Thalassemia	41 (22.0)	38 (20.4)	0.704	31 (20.4)	36 (16.4)	0.320
Heart	1 (0.5)	0 (0)	0.317	1 (0.7)	0 (0)	0.228
Multiparity	135 (72.6)	129 (69.4)	0.493	122 (80.3)	139 (63.2)	<0.001
History of macrosomia	4 (2.2)	4 (2.2)	1.000	3 (1.9)	2 (0.9)	0.381

*n (%), **Mean ± standard deviation (SD)
Abbreviations: GDM: gestational diabetes mellitus, PPW: pre-pregnancy weight, BMI: body mass index, PPBMI: pre-pregnancy BMI, TWG: total weight gain, GA: gestational age at birth.

TABLE 2. Analysis of maternal and fetal outcomes of GDM (n=372) and non-GDM (n=372).

	GDM*		*p*-value	Non-GDM *		*p*-value
	Obesity (186)	Non (186)		Obesity (152)	Non (220)	
PIH	26 (14.0)	7 (3.8)	0.001	14 (9.2)	6 (2.7)	0.006
GHT	13 (7.0)	1 (0.5)	0.001	6 (4)	3 (1.4)	0.111
Preeclampsia	13 (7.0)	6 (3.2)	0.099	8 (5.3)	3 (1.4)	0.029
C/S	64 (34.4)	57 (30.7)	0.439	58 (38.2)	64 (29.1)	0.067
PPH	6 (3.2)	5 (2.7)	0.760	3 (2.0)	6 (2.7)	0.642
FBW (Kg)**	3.3±0.5	3.1±5.1	0.002	3.6±0.4	3.3±0.5	<0.001
SGA	3 (1.6)	9 (4.8)	0.078	1 (0.7)	3 (1.4)	0.516
LGA	75 (40.3)	54 (29.0)	0.022	121 (79.6)	101 (46.0)	<0.001
NICU	23 (12.4)	20 (10.8)	0.627	21 (13.8)	10 (4.6)	0.
RD	25 (13.4)	21 (11.3)	0.529	16 (10.5)	13 (5.9)	0.103
Mild	21 (11.3)	20 (10.8)	0.869	16 (10.5)	11 (5.0)	0.043
Moderate	4 (2.2)	1 (0.5)	0.177	0	2 (0.9)	0.239
Preterm	13 (7.0)	24 (12.9)	0.057	2 (1.3)	11 (5.0)	0.057
Fetal anomaly	21 (11.3)	23 (12.4)	0.748	12 (7.9)	16 (7.3)	0.823
Birth injury	1 (0.5)	3 (1.6)	0.315	4 (2.6)	5 (2.3)	0.825
Hypoglycemia	8 (4.3)	5 (2.7)	0.397	2 (1.3)	1 (0.5)	0.361
Neonatal Jaundice	16 (8.6)	19 (10.2)	0.594	12 (7.9)	11 (5.0)	0.254
Birth asphyxia	4 (2.2)	1 (0.5)	0.177	3 (2.0)	0	0.036

*n (%), **Mean ± standard deviation (SD)

Abbreviations: GDM: gestational diabetes mellitus, PIH: pregnancy induced hypertension, GHT: gestational hypertension, C/S: primary cesarean delivery, PPH: postpartum hemorrhage, FBW: fetal birth weight, SGA: small for gestational age, LGA: large for gestational age, NICU: neonatal intensive care unit admission, RD: respiratory distress.

Lyu reported from China in year 2023 and 2024 that obese mothers had lower TWG than their non-obese counterparts.[14,15] TWG during pregnancy came from maternal and fetal components. Maternal factors, namely insulin level and glucose level, played important roles during the first half of pregnancy. Fetal factors, namely fetal growth and fluid expansion, caused the weight gain in the latter half of pregnancy.[20] From Lyu's study, obesity and GDM were independent factors for TWG. The findings from the current study supported Ke's and Lyu's findings.

Previous literature reported that pre-pregnancy overweight/obesity, GDM and excessive weight gain were independent factors of LGA risk.[14,15] Theoretically, obesity results from food overconsumption. Pregnant women tended to increase their food consumption due to high levels of progesterone during pregnancy.[21] However, pregnant mothers with obesity were subjects of concern to their attending physicians. Diet control and GDM screening were major interventions.

From the current study, obesity was a risk factor for PIH development among pregnant women. Insulin resistance and low-grade inflammation of endothelium and increased sodium resorption were the explanation of PIH among obese subjects.[5] Chinese studies by Sun and Ke reported that obesity among GDM subjects exhibited higher risk of PIH than those with normal weight.[14,22] A report from the US by Kim in 2016 said participants with obesity had four-fold increased risk of GHT than those with normal-weight pregnancies.[23]

From the present study, obesity brought an increased risk of fetal birth weight consequences to heavy weight babies (LGA or macrosomia) in both GDM and non-GDM groups. This finding supported results from previous studies that obesity was associated with higher neonatal birth weight and macrosomia.[13-16,22,23] Risk of higher neonatal birth weight from maternal obesity ranged between 1.7 and 2.8 fold.[13-16,22,23] Excessive nutritional environment, hyperlipidemia and high blood glucose levels were common findings among obese women. Fetuses in obese pregnant women were also exposed to high levels of glucose leading to higher fetal insulin secretion, fetal overgrowth and adipose tissue deposition.[5,15]

From the current study, obese subjects had a higher risk of preeclampsia than normal-weight pregnant mothers among non-GDM groups. However, there were no significant differences between obese and non-obese subjects in pre-eclampsia development among the GDM group.

Lyu and colleagues reported in 2024 that obesity and GDM were not associated with pre-eclampsia occurrence.[15] However, Lyu used the BMI cut point at a level equal to or more than 28 kg/m^2, while the current study used the BMI cut point at a level equal to or more than 25kg/m^2 according to the South and Southeast Asia consensus suggested for the Thai population.[19] Data from Sweden and China in 2021 said that risk of severe preeclampsia from obesity in Swedish and Chinese subjects were 2.1 and 4.5 fold, respectively. Severe pre-eclampsia's risk from GDM in both populations were 2.3 and 2.1 folds, respectively.[24] Ke's study reported from China that high weight and obesity were prognostic factors for pre-eclampsia development with OR at 2.7.[14] The association of obesity and GDM to pre-eclampsia was inconsistent among previous studies. Prevalence of pre-eclampsia in the current study, Lyu's, Ke's, Yang's and Swedish studies were 2.6, 1.6, 5.5, 1.6 and 0.9 percent, respectively.[14,15,24]

Pathophysiology of pre-eclampsia was a consequence of vascular factors (endovascular trophoblast invasion of uterine spiral arterioles), immunological factors (dysfunction between maternal, placenta, and fetus tissue), inflammatory and genetic factors.[25] Obesity and GDM might be associated with pre-eclampsia. Most obese subjects had metabolic syndromes consisting of insulin resistance.[5] Consequences of insulin resistance were low-grade inflammation, vascular endothelial activation and increased sodium reabsorption.[25]

From the current study, subjects with obesity in the non-GDM group had an increased risk of NICU admission, mild RDS and birth asphyxia in neonates. Addicott from the US reported in 2024 that increased maternal BMI brought a higher risk of adverse neonatal outcomes, namely preterm, low Apgar score, prolonged neonatal length of stay and NICU admission than those with normal-BMI.[26] Lutsiv from Canada reported in 2015 that morbidly obese patients (Class III, BMI ≥ 40kg/m^2) had higher risk of adverse neonatal outcomes than patients with class I and class II obesity (BMI 30 to 39.9kg/m^2).[27] Data from China and the Philippines in 2016 and 2017 showed that high BMI of patients was associated with poor adverse neonatal outcomes.[23,28]

Overconsumption of food resulted in obesity. Increased adipokines and cytokines in obese subjects led to chronic inflammation. This might be an explanation of adverse neonatal outcomes with obese mothers.[5] The findings from the current study supported existing literature.[23,26-28]

Among GDM groups, obese subjects had higher risk of GHT than non-obese subjects from the present study with statistical significance as shown in Table 2. From US and Chinese studies, overweight and obese participants had significantly higher risk of GHT than their normal-weight counterparts.[22,23] The findings from the current study are in lieu with US and Chinese studies only in GDM subjects. Another explanation was that the average BMI of the GDM group was higher than the non-GDM group (25.7 vs 24.5, p-value 0.004). This indicated that subjects with GDM were more likely to be obese than their non-GDM counterparts. Data from the previous studies were summarized and presented in Table 3.

The study's strengths include its large sample size, robust methodology, and comparison between GDM and non-GDM groups. The retrospective nature is a notable limitation, as the study relies on historical medical records, which may have incomplete data. The lack of consideration for lifestyle factors (diet, exercise) and genetic predisposition to obesity is also a limitation. To prevent the risk of adverse pregnancy outcomes caused by obesity, the following suggestions should be considered namely weight reduction before pregnancy, proper pre-pregnancy weight, pre-conceptional counseling, early ANC and suitable plans should be made and agreed with pregnant women.

CONCLUSION

Obesity in pregnant women (BMI equal or more than 25 kg/m^2) increased the occurrence of PIH, FBW and LGA when compared to those with normal weight. Moreover, obese pregnant women concurrent with GDM had higher risk of GHT. Whereas obese pregnant women with non-GDM had increased risk of preeclampsia, NICU admission, RD, and birth asphyxia.

Gestational diabetes mellitus (GDM) is a common

TABLE 3. Comparison adverse maternal and fetal outcomes of obese women.*

Authors	Present	Yue S.	Alfadhli	Ke JF.	Lyu Y.	Kim SS.	Sun Y.	Yang Y.	Yang Y.	Addicott	Kim T.	Lutsiv
Years	2024	2022	2021	2023	2024	2016	2020	2021	2021	2024	2017	2015
Country	Thailand	Vietnam	Saudi Arabia	China	China	US	China	Sweden	China	USA	US	Canada
Total case (N)	744	4,970	531	746	802	112,309	3,172	555,446	79,243	858	338	-
Total Obese	338(45.4)	267(5.4)	254(47.8)	218(29.2)	71(8.9)	19,482	59	63,846	2,693	472	338	-
GDM	186(55)	78(29.2)	170(66.9)	218	23(32.4)	1,928(1.7)	12	-	-	59	44(13)	-
cut-off BMI	≥25	≥27.5	≥30	≥24	≥28	≥30	≥28	-	-	≥30	≥30	≥30
PPBMI(kg/m^2)**	29.9	-	35.6	25.8	-	18.5	-	24.6	21.9	40.1		-
Age (yr)**	31	30.3	32.9	31	30.6	27.8	18-44	30.9	28.6	27.1	29	-
GA (weeks)**	38.5	38.6	-	38.6	48	-	-	-	-	38.9	38.5	-
Multiparity	257(76)	167(62.5)	-	99	42	13,473	-	-	-	39	250(74)	-
Hx.macrosomia	7(2.1)	12(4.5)	-	-	-	-	-	-	-	-	-	-
PPH	NS	-	-	-	NS	NS	-	-	-	-	S	S
GDM	-	-	-	-	-	S	S	-	-	S	S	S
TWG (kg)	S	S	-	S	-	-	-	-	-	S	-	S
PIH	S		-	S	-	-	-	-	S	-	-	S
GHT	S	-	-	-	-	S	S	-	-	-	NS	S
Preeclampsia	S	-	-	NS	NS	-	-	S	-	S	NS	S
C/S	NS	S	S	S	S	S	S	S	S	S	S	S
FBW (Kg)	S	-	S	S	S	S	S	-	-	-	-	S
SGA	NS	-	-	NS	NS	-	NS	S	-	-	-	NS
LGA	S	S	S	S	S	S	S	S	S	-	S	S
NICU	S	-	S	-	-	S	-	-	S	S	S	S
RDS	S	-	-	-	-	S	-	-	S	-	S	-
Preterm	NS	S	-	S	NS	S	NS	S	-	S	NS	S
Birth asphyxia	S	-	S	-	-	NS	-	-	-	NS	S	NS

*n (%), **Mean ± standard deviation (SD)

Abbreviations: GDM: gestational diabetes mellitus, PPBMI: pre-pregnancy BMI, GA: gestational age at birth, Hx.macrosomia: History of macrosomia, PPH: postpartum hemorrhage, TWG: total weight grain, PIH: pregnancy induced hypertension, GHT: gestational hypertension, C/S: primary cesarean delivery, FBW: fetal birth weight, SGA: small for gestational age, LGA: large of gestational age, NICU: neonatal intensive care unit admission, RDS: respiratory distress syndrome.

complication during pregnancy. Maternal hyperglycemia leads to fetal hyperglycemia and stimulates excessive somatic fetal growth causing macrosomia and large-for-gestational age (LGA) neonates. Maternal obesity is also the most common metabolic complication in pregnancy. Normoglycemic obese women had an increased risk of macrosomia and higher rate of cesarean delivery.

Obesity in pregnancy increased higher occurrence of PIH, FBW and LGA than those with normal weight among pregnant women. Obese pregnant women with GDM had more GHT than non-obese pregnant women. Obesity with non-GDM pregnant women increases the risk of pre-eclampsia, NICU admission, RDS, and birth asphyxia

Data Availability Statement

Data about individual identified samples of this research will be available from the corresponding author Piyawan Pariyawateekul upon reasonable request after the main results of the research have been published.

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DECLARATIONS

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Conflict of Interest

There was no conflict of interest.

Registration Number of Clinical Trial

Not applicable.

Author Contributions

Conceptualization and methodology, P.M., P.P. and B.S.; Investigation, P.M, P.P, M.P; Formal analysis, P.M., P.P., M.P., and B.S.; Visualization and writing – original draft, P.M., P.P., K.S., and K.B; Writing – review and editing, P.M., P.P., W.L., and M.P; Funding acquisition, P.P.; Supervision, P.P., and K.S. All authors have read and agreed to the final version of the manuscript.

Use of Artificial Intelligence

No content was generated using AI.

Institutional Review Board Statement

This retrospective review has been approved by the local Institutional Review board (reference number: 77/66).

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